As to the population of the Libby area, Peipins et al (2003), p.1758, speaks of a population of "9521 persons in Central Lincoln County, a population that has been relatively stable for the past 30 years (U.S. Bureau of the Census 2002)." Peipins et al (2003) discusses the demographics of study participants and discusses the results of screenings administered by the ATSDR in 2000 and 2001. Chest x-rays were taken on 6,668 participants, representing 61% of the area population, of whom 18% (1,186 of 6,668) had pleural abnormalities. This is a very large number for a small community.

Peipins et al (2003), Table 3, notes the exposure pathways for the 1,186 with pleural and/or interstitial abnormalities:

Ever worked for W.R. Grace	186
Lived with W.R. Grace workers	358
Environmental only exposures derived as 1186-358-186=	642
Resident (more than 6 months before 1991)	1,186.

Note that all participants are "residents." Peipins et al (2003), p.1755, states "the pathways presented here are not mutually exclusive." Many community exposure pathways are listed. Most study participants with abnormalities are not workers or family members of workers, but are simply residents of the Libby area.

The pattern seen in exposure histories for patients of the CARD Clinic is consistent with the findings in Peipins et al (2003). Most patients are

community members who are not workers or family members of workers. Indeed the percentage of new patients who are community members has increased in recent years. An important aspect of Libby area exposures is that residents had a 24 hour exposure, with no respite away from exposure to clear out the airways. This appears to be generally different from exposures reported elsewhere.

The CARD Clinic has diagnosed over 1,800 patients with asbestos related disease by either plain chest x-ray or CT scan, and has confirmed diagnoses on about 100 patients in other areas of the U.S. All had exposures to Libby asbestos due to W.R. Grace or Zonolite Company operations in or near Libby, Montana. Very few patients had exposures to other asbestos outside the Libby area. Few patients had non-vermiculite related asbestos exposures within the Libby area. All patients in the over 1,800 diagnosed above have asbestos disease due to exposure to Libby asbestos with its source in W.R. Grace and Zonolite Co. mining and other activities.

36. The following numbers are supplied by the McGarvey, Heberling, Sullivan & McGarvey law firm. Of 653 Libby Claimants who are patients of CARD:

Ever worked for W.R. Grace	189	29%
Subcontractors at W.R. Grace	26	4%
Family household member of W. R. Grace worker	143	22%

Community members (all others)	295	45%
Total	653	100%

See printout, Exh. 4 "Client Sort by Exposure (Community, Family Member, Worker)" (CARD patients only) 5/28/08. While these numbers are representative only of Libby Claimants, they are consistent with the findings in Peipins et al (2003), in that the greatest number are community members, who typically played baseball near Grace operations, played as children on piles of Grace vermiculite, or as residents breathed in asbestos in the dust in the air in the Libby area.

37. In addition to the vermiculite contamination widespread in the Libby area, a contributing factor in the widespread pleural disease is the minimal exposures which triggered disease. There are many examples in a patient cohort of surprisingly minimal exposures which led to significant disease:

> Tom Murray, a former federal magistrate judge, worked at the mine during the summers of 1948 and 1949, and died of asbestos lung cancer.
>
> Betty Maxwell who only visited Libby for 1-2 week periods for about ten summers, and cleaned cabins, now has severe asbestos disease and is on oxygen.
>
> Victoria Skidmore who only visited Libby for errands averaging about two trips per month for 14 years, now has pleural mesothelioma.

38. A hallmark of pleural disease from Libby asbestos exposure is that it is highly progressive. In Whitehouse (2004) (Exh. 2), 123 patients with

two or more sets of pulmonary function tests were studied. All had pleural disease, and 55% had no evidence of interstitial changes. There was an average of 35 months between first and last lung function test. 76% of the patients showed progressive loss of pulmonary function. The extent and rapidity of loss of pulmonary function were highly significant. Page 221 states that for the 123 patients "the average yearly loss was 2.2% for FVC, 2.3% for TLC, and 3.0% for DLCO." To my knowledge, no study of asbestos disease from predominately chrysotile exposure shows overall progression rates for lung function loss anything like those seen in Libby, where most patients have had relatively light exposures.

As of 3/11/09, 36 of the 123 patients in the study had died. For 30 of 36, asbestos related disease was a significant factor in death. Life expectancy at date of diagnosis was calculated for each of the 30. The average loss was 9.3 years of life expectancy for the 30 patients. See Exh. 5. "Dead in 123 patients in Whitehouse (2004)."

39. Interestingly, Alfonso (2005), an amphibole study from Australia, similarly finds an "average rate of decline of DLCO . . . [of] about 2.2% per year [0.55/24.8] in people with asbestosis." Asbestosis was considered present upon an ILO score of 1/0 or greater. DLCO decline was not investigated for pleural disease in the study cohort. In another amphibole study, Cookson (1983), abstract, states "[t]he ratio of transfer factor to

effective alveolar volume correlated directly with the degree of pleural thickening as alveolar volume fell with increasing severity of pleural disease." Sichleditis (2007) is a study of environmental exposure to tremolite asbestos in Almopia, Greece. It finds significant progressive lung function loss in patients with pleural disease. Villagers whitewashed buildings using mineral dust, which contained tremolite asbestos. A doubling of the size of pleural plaques was found in 126 subjects followed for 15 years. 18 of the 126 had pulmonary function tests in 1988 and 2003. Percent predicted for total lung capacity dropped from 96% to 76% (1.3% per year), forced vital capacity dropped from 95% to 80% (1.0% per year), and residual volume dropped from 98% to 66% (2.1% per year).

The 18 subjects with pulmonary function tests had comparatively mild radiographic progression, with only a 60% increase in area of pleural plaques (p.636) (data at Table 2, p.640). The authors report that "no obvious radiological abnormalities were detected in lung parenchyma," p.636.

40. Rapid progression in the Libby cohort can also be seen radiographically. Exhibit 6 is a Chart titled "Chart Case # Type of Progression," which presents a collection of 18 cases of rapid progression in seven years or less, secondary to pleural disease and/or interstitial disease. CDs are attached at Exh. 6 showing photos of pleural plaques and the progression of plaques to interstitial disease. To my knowledge,

progression of pleural disease of this nature has not been reported elsewhere.

41. Pleural disease from exposure to Libby asbestos appears to be far more severe than asbestos pleural disease reported elsewhere. The CARD mortality study presents 24 deaths by pleural disease with no interstitial disease on last chest x-ray. Exh. 7. ATS (2004) p.707, reports only five total cases of death by pleural disease outside Libby. I have read reports of others, but they remain very few. In Miles et al (2008), p. 2, the Australian authors report that death secondary to diffuse pleural thickening is "very rare." Commonly the mechanism for death by pleural disease is loss of pleural space resulting in episodes of hypoxia. Due to the highly progressive nature of Libby asbestos disease, once diagnosed with pleural disease, with multiple pleural plaques or diffuse pleural thickening and a loss of lung function, a patient has a high probability of progressive disease and early mortality due to asbestos disease.

42. The CARD mortality study observed that 59% (110/186) of deceased patients of CARD died of asbestos disease, meaning that asbestos related disease was at least a significant contributing factor in the death. See Exh. 7, Summary of Mortality Study Disease Percentages. Cause of death per death certificate was noted, and also a determination was made by best available information review of medical records, per Selikoff (1992).

In contrast, a patient diagnosed with asbestos disease from predominately chrysotile exposure has a much lower likelihood of death.

43. In the CARD pleural disease cohort, there is a large number of patients with severe pulmonary function impairment. To my knowledge, no similar incidence of impairment is reported in an asbestos pleural disease cohort elsewhere, or in a chrysotile disease cohort elsewhere. Note that some Libby patients have severe impairment mainly due to smoking. A proper cohort comparison would include cohorts with asbestos pleural disease or chrysotile disease including all smokers.

Per the records of the McGarvey, Heberling, Sullivan & McGarvey Law Firm, of 653 Libby clients with asbestos disease from exposure to Libby asbestos who are also patients of CARD, 77 are on oxygen, and 171 have at least one of the three main indicators of severity of asbestos disease under 60% of normal (FVC, TLC or DLCO). Exhibit 8. While these numbers are representative of the listed patients only, based on clinical observation, it is likely that they are indicative of severity of asbestos disease in the CARD patient cohort. (Miller norms are generally used for DLCO. If translated to Crapo, a decrease of roughly 10% of predicted occurs.)

A number of studies report an increased incidence of extensive fibrosis and pleural effusions associated with exposure to Libby asbestos, as compared to what is seen in other populations. See Lockey (1984),

McDonald (1986), Amandus (1987) and Libby Studies listed.

44. Attached is a chart titled "Mesothelioma Cases with Exposure to Libby Asbestos as a significant factor." (7/23/08). Exhibit 9 (and CD with data). 31 cases of mesothelioma are verified, where exposure to Libby asbestos was a significant factor in producing the mesothelioma. Three others await verification. Verification is per death certificate or pathology report, or both. This is an extremely high incidence of mesothelioma in a community with an average population of 9,521. Peipins et al (2003); ATSDR (2002). The background mesothelioma rate is thought to be about one per million per year. Roggli (2007). There have been 20 mesotheliomas with exposure to Libby asbestos as a significant contributing factor in the past 12 years (1996-2007), or 1.7 per year. If one uses the 20 mesotheliomas, then the Libby area, with a population of about 10,000 is at 166 per million per year, or 166x the background rate. If one uses the 10 mesotheliomas with the Libby area as residence at death, the rate is 83 per million per year, or 83x the background rate. Libby's mesothelioma rate is certainly the highest in the United States. The Fraser and Pare text, p.886, states the mesothelioma rate to be "33 per million per year for South Africa and to be 66 per million per year for Western Australia." Libby's mesothelioma rate is among the highest in the world. It is also noted that the crocidolite mine at Wittenoom, Australia closed in 1966, whereas the

Libby mine closed in 1990. The full manifestation of mesothelioma in Libby is far from completion.

45. Libby has also had 13 environmental exposure mesothelioma cases (1995-2007) where the exposure to Libby asbestos is considered to be a significant factor in producing the mesothelioma. Exh. 9. 11 cases were described in Whitehouse et al (2008). Two more have been verified since the date of submission of the article. "Environmental" excludes miners, but includes immediate family members of miners. Two mesothelioma cases are household family members of workers at the mine (Orem and Flatt). If one uses 11 environmental cases 1995 -2007, this could translate to 0.85 per year (11 cases in 13 years), or 85 Libby environmental mesothelioma cases per million per year. However, one is alive and only five of the 11 died as Libby residents. Many Libby patients in end stage disease move to areas with major medical centers. If we use only the five who died as Libby area residents, then the rate is 38 per year, or 38 per million per year. Libby's environmental mesothelioma rate is the highest in the United States, and is among the highest in the world.

Berry (1996), "Mesothelioma Incidence and Community Asbestos Exposure," presents mesothelioma statistics for workers and community residents near the Johns-Manville Plant in the City of Manville, Somerset County, New Jersey. The Manville plant operated 1912-1980. The Libby

mine and mill operated from the 1930s to 1993. Even though the Libby mine operated for fewer years and closed 13 years later, such that the fulminate presentation of mesothelioma cases is yet to occur, the Libby community mesothelioma rate appears to be higher than that of Somerset County, New Jersey.

The Manville plant "employed up to 3,500 people," p.34. The Libby mine and mill employed at most about 160 people, a difference of about 22x. The town of Libby has had a population of about 3,000. The town of Manville, situated adjacent to the plant, had an average population of 10,923, p.36. Central Lincoln County (defined as a 10 mile radius from the center of Libby) had 9,541 persons, Peipins (2003). Somerset County, New Jersey, had an average population (1980 and 1990) of 208,435. This is about 22x the size of Central Lincoln County. So, whereas the Manville workforce was about 22x of that of Libby, the community size from which mesothelioma cases is drawn is also about 22x that of Libby.

Berry (1996), shows total mesothelioma cases for 1979-1990 for Somerset County at 143. 61 plant employee cases are subtracted out, for a net community case number of 82. Using 82 community mesothelioma cases in 208,435 residents over 11 years results in 36 per million per year. Using 11 Libby community mesothelioma cases in 9,541 residents over 11 years (1995-2006), results in a rate of 105 per million per year.

Berry (1996), p.36, used "address at time of diagnosis" to determine eligibility. If we apply this to the Libby community mesothelioma cases, then we find five with Central Lincoln County residence at the time of diagnosis. Using these five cases results in a mesothelioma rate of 48 per million per year (5/11 x 105), exceeding Somerset County, New Jersey at 36 per million per year.

Berry (1996), p.38, states that particulate from the Manville plant "regularly coated cars, homes and yards like a fresh snowfall in the immediate community." The Libby mine and mill were seven miles northeast of Libby, with a prevailing wind from the west and southwest. The entire production of the mine was shipped out of the Libby railroad yard in town, and W.R. Grace had a small expansion plant and bagging plant near the railroad tracks. Libby residents report substantial dust in the town, but it appears that the Manville asbestos dust concentrations in town were significantly greater. However, the Libby asbestos is amphibole asbestos, whereas Manville used approximately 95% chrysotile asbestos, p.38. Amphibole asbestos is more toxic, which likely accounts for the greater rate of mesothelioma cases in the Libby community, even though exposures were not as heavy.

Berry (1996), notes that due to insufficient exposure histories, there could be no sorting of community (non-worker) cases into household

member of worker cases and pure environmental cases. This sort can be accomplished with the Libby community mesothelioma cases.

46. Sullivan (2007), Table 1, conservatively finds as of 2001, 154 deaths among Libby mineworkers from asbestos related disease (99 lung cancers, 15 mesotheliomas and 40 asbestosis). Sullivan (2007), Table 2, also finds 111 miner deaths by non-malignant respiratory disease (NMRD). This is a larger catchall category, and includes the 40 asbestosis deaths. In all likelihood, if the Selikoff (1991) best available information analysis were applied, many of the other 71 NMRD deaths in Libby miners could be identified as due to asbestos related disease. The Sullivan (2007) count of 154 miner deaths due to asbestos disease is very conservative.

47. The CARD mortality study adds another 31 miners (including subcontractors) who have died from asbestos related disease since 2001. Exh. 7 (as of 7/9/08). In addition to the subjects in the mortality study, 23 more workers' (including two subcontractors) death certificates and records have been reviewed by me (Exh. 10 as of 3/12/09). These 23 ARD deaths were persons not seen at CARD or by me in Spokane. The total for workers is 208 (154 + 31 + 23).

The CARD mortality study shows 73 family members or community residents died of ARD. Exh. 7. In addition to the subjects in the mortality study, 14 more family and community members' death certificates and

records have been reviewed. Exh. 10. These 14 ARD deaths were persons not seen at CARD or by me in Spokane. The total for family and community members is 87 (73 + 14). The above conservatively totals 295 (208 + 87) deaths in the Libby cohort due to asbestos-related disease, as of 7/9/08.

Studies on deaths in the Libby cohort are summarized on the attached chart "Libby Cohort Deaths per Source," Exh. 21. It also appears that W.R. Grace had documented 30 lung cancers as of 1985. See Exh. 22, memo of 9/17/85.

48. "Lincoln County, Montana, had the highest age adjusted asbestosis mortality rate in the United States for 1988-1997. (Castellan R. unpublished data)," letter by Peipins et al in Environmental Health Perspectives 112:a83. See also Whitehouse (2004), p.224. It appears that this statement applies to 1998-2007 as well.

ATSDR (2002), "Mortality in Libby, Montana 1979-1998," is a death certificates study. Some results from ATSDR (2002) were published in Horton et al (2006), "A Review of the Federal Government's Health Activities in Response to Asbestos-Contaminated Ore found in Libby, Montana." ATSDR (2002), p. 1, states the following conclusions:

> For the 20 year period reviewed in this report (1979-1998), mortality in Libby resulting from asbestosis was 40 to 80 times higher than expected. Mortality from lung cancer was also elevated, with a 20 to 30 percent excess over this time period.

Table 10 shows 12 deaths in category 501 "asbestosis," resulting in standardized mortality ratio 65x higher than that of the U.S. reference population.

ATSDR (2002) is a very conservative study, since it apparently used only the primary cause of death, not underlying or contributing causes, and only counted those who were Libby residents at death. Libby's end stage patients often move to larger towns with major medical centers, and die there. ATSDR (2002) may have understated the incidence of asbestosis deaths in the Libby area. It is not clear how the U.S. reference population incidence of asbestosis was calculated, and whether only the primary cause of death was used for the U.S. reference population.

We will apply the ATSDR (2002) approach of using only the death certificates' primary cause of death, to the data in the CARD mortality study. We also include only those who died as residents of the "Libby area," defined as "Central Lincoln County," per ATSDR (2002). For the ten years 1998-2007, the result is 10 for category 501 "asbestosis." Exh. 7. We double the ten year total for comparison of the ten year rate to the ATSDR (2002) 20 year U.S. baseline rate. This assumes that the baseline U.S. death rate for asbestosis has not significantly changed from that used in ATSDR (2002). (1998-2007: 10 asbestosis deaths x 2 = 20. 65 x 20/12 = 104). It appears that the death rate for 1998-2007 is 104x the U.S. rate. The Libby area has

the highest asbestosis mortality in the United States.

49. There is a latency period between exposure and the first appearance of asbestos disease on chest x-ray or CT. During the latency period, microscopic asbestos fibers are working at a microscopic level, until they become detectible on chest x-ray or CT. ATS (2004), p.695, suggests a minimum latency period of 15 years. Asbestos pleural disease from exposure to Libby asbestos often appears radiographically in less than what would be considered a minimum latency period of 10-15 years. We find no report of short latency in disease from predominately chrysotile exposures.

Data on Libby asbestos disease indicates that changes of asbestos disease can occur in as little as 3-5 years (clearly defined plaques), whereas chrysotile latency periods are generally over 10 years. With Libby asbestos, the range generally appears to be about 5-40 years, with an average latency period of about 15-30 years from first exposure to diagnosis. Miles et al (2008), p. 3, report that "DPT can develop within a year from exposure to asbestos." We have not seen a latency that short in cases of Libby exposure.

The results of W.R. Grace's annual chest x-ray program for workers from 1959 to 1980 appear to confirm that the Libby asbestos has a very high toxicity. Grace's in-house studies in 1969, 1975 and 1976 on annual x-rays